A Dissertation on

OPC POISONING AND SERUM CHOLINESTERASE LEVELS

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600032

In partial fulfilment of the Regulations
for the Award of the Degree of

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
STANLEY MEDICAL COLLEGE
CHENNAI – 600 001

APRIL 2017

CERTIFICATE BY THE INSTITUTION

This is to certify that **Dr.M.OWAISE AHMED,** Post - Graduate Student (May 2014 TO April 2017) in the Department of General Medicine STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on "**OPC Poisoning And Serum Cholinesterase Levels"** under my guidance and supervision in partial fulfillment of the regulations laid down by the Tamilnadu Dr. M. G. R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in April 2016.

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DECLARATION

I, **Dr.M.OWAISE AHMED,** declare that I carried out this work on **"OPC Poisoning And Serum Cholinesterase Levels"**at the out patient department and Medical wards of Government Stanley Hospital . I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

This is submitted to The Tamilnadu DR. M. G. R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M. D. Degree examination in General Medicine.

Dr.M.OWAISE AHMED

ACKNOWLEDGEMENT

At the outset I thank our dean **Dr. ISAAC CHRISTIAN MOSES, M.D,FICP, FACP.,** for permitting me to carry out this study in our hospital.

I express my profound thanks to my esteemed Professor and Teacher **Dr.P.VASANTHI, M.D.,** Professor and HOD of Medicine, Stanley Medical College Hospital, for encouraging and extending invaluable guidance to perform and complete this dissertation.

I immensely thank my unit chief **Dr.RAJAN, M.D.,** for his constant encouragement and guidance throughout the study.I would also like to extend my heartfelt thanks to my former unit chief **Dr.SOUNDARARAJAN, M.D.,** for her guidance in completing this dissertation.

I wish to thank **Dr.KARUNAKHARAN,M.D.,** and **Dr.RAVICHANDRAN, M.D**, Assistant Professors of my unit, Department of Medicine, Stanley Medical College Hospital for their valuable suggestions, encouragement and advice.

I sincerely thank the members of Institutional Ethical Committee, Stanley Medical College for approving my dissertation topic.

I thank all my colleagues, House Surgeons, and Staff nurses and other para medical workers for their support.

I sincerely thank all those **patients** who participated in this study, for their co-operation.

Above all, I thank the **Almighty** for gracing me this opportunity, health, and knowledge throughout this study.

CONTENTS

OPC POISONING AND SERUM CHOLINESTERASE LEVELS

INSECTICIDES-OPC AND CARBAMATES

INTRODUCTION

OPC poisions are esters of phosphoric acid and form two series of compounds.

Organophosphorus compounds and carbamate are two groups of cholinesterase inhibiting pesticides that commonly produce human toxicity.

Those cholinesterase-inhibiting (anti- cholinesterase) insecticides that contain phosphorous will be collectively termed organic phosphorus compounds.

Those that contain the OC - ON linkage will be termed carbamates.

REVIEW OF LITERATURE

Anticholinesterase insecticides likely kill more people each year than acute poisoning by any other chemical. Globally, severe occupational, unintentional poisoning also happens where such insecticides are used.

Organophosphorus compounds were initially developed 95 chemical warfare agents. They were recognized after World War II. More than 50 highly effective organophosphorus compounds are in use presently.

In industrialized countries access to toxic pesticides as much more controlled. Hence Anticholinesterose poisoining occurs less commonly.

In rural India poisoning occurs regularly and might result in hundreds of casualities being treated by clinicians with little experience of this potentially lethal toxic syndrome.

World health Organisation classification of pesticides:

Class la extremely hazardous

Class lb Highly hazardous

Class li Moderately hazardous

Class III slightly hazardous and active ingredients unlikely to present acute hazard in normal use.

OLDER CLASSIFICATION:

Alkyl compounds include	hexaethyl tetraphosphates
	Tetraethyl pyrophosphate
	Octamethyl pyrophosphamide
	Malathion
	Cystox
	Dipterex
Aryl compounds include	Parathion
	Para-Oxon
	Methyl Parathion
	Chlorothion
	Diazinon

MODERN CLASSIFICATION:

Agirculture insecticides (highly toxic)

- OMPA
- TEPP-Parathion
- Phosdrin
- Didyston

Animal insecticides (Moderately toxic)

- Ronnel
- Coumephos
- Trichlorfon

House held insectices (low toxic)

- Malathion
- Diozinon

Victims are usually children, farmers and unskilled labourers.

Typically patients present following unintentional or suicidal ingestion of anticholinesterose insectices or after working in areas recently treated with these compounds.

Direct dermal contact with I certain types of these insecticides may be rapidly poisonous.

PHARMACOLOGY:

The basic formula for cholinesterase inhibiting compounds.

X represents leaving grcup

R1 u1 R2 my be aromatic cr aliphatic groups

Group 1 compounds contain a quarternary nitrogen at the X-position and are collectively termed phosphoryl cholines.

Group 2 compounds are called flucrophosphates because they possess a fluorine molecule as the leaving group.

Group 3 compound is a cyanide molecule or a halogen other than fluorine. The most well known agents in this group are cyonophosphates such as the chemical weapon tabun.

The fourth group is the broadest and comprises various subgroups based on the configuration of the R1 and R2 groups with majority falling ino the category of either a dimethoxy on diethoxy compound.

Most of the insectices in use today fall into this last class.

Direct acting up insecticides inhibit acetylehelinesterose without being metabolized in the body.

Indirect acting or insecticides such as parathion and malathion require partial metabolism to become para oxon and molaoxon respectively to become active.

The organophosphates bind to a hydroxyl group at the active site of Ach enzyme.

As the leaving group of the OP insecticide is split off by ache stable but reversible bond results between the remaining substituted phosphate of the orgnophosphate and Ache, effectively inactivating the enzyme.

They act via all routes including

Transdermal

Trans conjunctival

Inholational across the gastrointestinal

and genitourinary mucosa and

through diurect injection.

ABSORBTION, FATE AND EXCRETION

Organophosphorus compounds are well abscobed through the mucus membrane of GIT, RS and intact skin.

Parothion is stored in the body fat and is slowly released in th circulation, prolonging the duration of its toxic action.

It is first metabolized into para oxon, which is the active toxic agent and then to paranitrophenal, which is excreted through urine.

Malathion is metabolized in the liver by the esterases. A part of the metabolized product is exrerected in urine as phosphate.

Parathion may be retained for a period of about a week.

Malathion for a period of more than one week.

The presence of broken skin, dermatitis and higher environmental temperatures further enhances cutoneuus absorption.

Most Ops are lipophilic and are therefore predicted to have a large volume of distribution and to rapidly distribute into tissue and fat where they are protected from metabolism.

Dimethoate, methamidophos and oxydemeton methyl are the three common ops that are not lipophilic with predicted small volumes of distribution and high plasma concentration.

Moe lipophilic are the compounds mere likely to cause cholinergic crisis.

PATHOPHISIOLOGY

Acetyl choline is a neurotransmitter found at both parasympathetic and sympathetic ganglia.

Skeletal neuromuscular junction.

Terminal junctions of post ganglionic parasympathetic nerves, post ganglionic sympathetic glands and at some nerve endings within the CNS.

Ach helps in transmission of impulses in the synaptic and neuro muscular junction.

As the axon terminal is deparised, vesicles containing Ach fuse with the nerve terminal, releasing Ach into the synapse or neuromuscular junction.

Ach then binds to post synaptic receptors leading to activation.

Activation alters the flow of Na;k; cr on nerve cells and alters membrane, resulting in propagation of the action potential.

Ops and Carbomates are inhibitors of carboxylic ester hydrclases within the body including

Acetyl cholinesterase

Buftyl cholinesterase

Hepatic carboxylesterases

Poraoxonases

Chymotrypsin

And other non specific proteases

Organophosphates irreversibly bind to cholinesterase, causing the phosphorylation and deactivation of acetylcholinesterase.

This results in accumulation of acetylcholine at the neural synopse causing on initial overstimulation, followed by eventual exhaustion and disruption of post synaptic neural transmission in the central nervous system.

Ache is found in human nervous tissue and skeletal muscle and on erythrocyte cell membranes.

Butryl cholinesterase is a hepatic derived protein that is found in human plasma, liver, heart, pancreas and brain.

Inhibition of Ach is thought to account for all or majority of clinical features.

CLINICAL MANIFESTATION:

Clinical findings of acute toxicity from OPS derive from excessive stimulation of muscorinic and nicotinic cholinergic receptors by Ach in the central and autonomic nervous system and at skeletal neuro muscular junction.

The classically described patient with severe of puisuning is one who is unresponsive with pinpoint pupils-muscle fasciculoticans, diaphoresis, salivation, lacrimation, urinary incontinence, diarrhea, emesis and an odonr of gastric or solvents.

The timing of onset of symptoms varies according to the route, degree of exposure and particularly the OP.

The speed of onset will also be affected by the quantity ingested and toxicity of the op.

Lipid solubility also likely affects time of OA set

Patients suffering massive ingestins can become symptomatic as quickly a 5 minutes following ingestion.

Oxon ops are already active on exposture and patients become symptomatic very soon after ingestion.

Some thion OPS are very rapidly converted to axons and can similarly produce symptoms rapidly.

In contrast patients ingesting thions that are slowly converted to active axons may not show symptom for hours.

Cholinergic receptors are found in both sympathetic and parasympathetic nervous system.

Hence the effects of excessive ach on autonomic nervous system may be variable.

Excessive muscarinic activity can be characterised by several mnemonics including

SLUDGE - Salivation

Lacrimation

Urination

Defecation

Gastrointestinal emedies

DUMB BELS Defecation

Urinatich

Micsis

Brenchospasm

Branchorrhoea

Emesis

Lacrimotion

Salivation

Miosis is the most consistently enccuntered sign. Brenchorihoea can be so prefuse that it mimics pulmenory oedema.

Cardiovascular monifeslations include mixed effect on the autonomic nervous system (including increased sympathetic tone) together with the consequences of induced hypoxia and hypovolemia

Admission heart rate is usually normal with relatively few patients expressing a tachycardia or bradycardia patients who have received Atropine before admission may be tachycardic.

Hypotension may occur because of stimulation of vascular receipts by excessive circulating Ach, severe volume los or myocardial dysfunction.

Respiratory complication of poisoning include.

(1)Direct pulmonory effect of bronchorrhoea, broncho constrction (s) neuro muscular junction failure in diaphragm and intercostal musle, loss of central respiratory drive.

Both bronchorrhoea and bronchoconstrictrs respond to adequate atrcpine therapy. Unfortunately neither neuromuscular junction failure nor loss of central respiratory drive respond to Atropine and patients must be intubated a ventilated till respiratory function returns.

Delayed syndromes:

The delayed syndromes include

(1) Neuromuscular junction dysfunction
(2) Organic phosphorus compound induced delayed neuropathy

Neuromuscular junction dysfunction:

A syndrome of delayed muscle weakness without cholinergic features op fasciculation resulting in respiratory failure.

The syndrome is defined as occurring 24 to 96 hours after acute of poisoining and after resolution of the cholinergic crisis.

Patients develop proximal muscle weakness especially of the neck flexers and cranial nerve PALSIES and progress to respiratory failure that may lAst for several weeks.

Consciousness is usually preserved.

Although the exact pathophysiologyof the syndrome is unknown, it is clearly due to dysfunction of the neuro muscular junction with respiratory failure resulting from weakness affecting the diaphragam and intercostal muscles.

The treatment of intermediate syndrome is supportive with airway protection and mechanical ventilation.

Pralidoxime or Atropine is not effective in the treatment of this disorder, although patients may require these medications to control concurrent cholinergic symptoms.

The weakness and paralysis commonly resolve in 5 to 18 days.

Organic phosphorus compound-induced delayed neuropathy.

Organophosphate induced delayed polyneurepathy is a rare complication that usually occurs 2-3 weeks, after acute exposure.

It is a mixed sensory/motor polyneuropathy affecting long myclinated neurons.

It appears to result from inhibition of enzymes other than Ache

Early clinical features are muscle cramps followed by numbness and paraesthesiac proceeding to flaccid paralysis of lower and subsequently upper limbs.

Paralysis of lower limbs is associated and foot drop and a high stepping gait-progressing to paraplegia.

Paralysis of the arms lead to wrist drop.

Sensery loss may also be present but is variable.

There is no specific therapy for OPIDN.

Delayed neuropathies are not usually associated with carbamate insecticides.

DIAGNOSTIC TESTING:

The most appropriate laboratory tests for confirming cholinesterase inhitition by insecticides are tests that measure.

1)Specific insecticids and active metabolites in blologic tissues.

2)Cholinesterase ativity in plasma or blood.

Verifying cholinesterate inhibites poisoning currently relies on measurement of cholinesterase activity.

Although urine and serum assays or op compounds and their metabolites are available, such testing is rarely obtainable within hours

Moreover normal ranges and toxic concentrations are not established for most compounds.

The two cholinesterases commonly measured are butyrylcholinesterase and red cell acetyl cholinesterase.

Blood samples for cholinesterase activity must be obtained in appropriate blood tubes.

Tubes containing flucrides will permanently inactivate the enzyme yielding falsely low activities and should never be used.

Specimens for RBC Ache are usually drawn into tubes containing a chelating anticoagulant such a EDTA to prevent clot formation.

Buche does not require on anticoagulant and can be drawn into tubes without chelators or anticoagulents.

ATROPINE CHALLENGE:

An atropine challenge may be helpful in diagnosing cholinergic poisoning in a patient who presents with findings suggestive of this disorder but in whom no history is available to suggest exposure to an op or carbomate insecticide.

In such individuals, a test dose of 1 mg of atropine in adolescent or adults should produce classic antimuscorinic findings in particular tachycardia, mydrasis and dry mucus membrane.

Conversely the persistence of cholinergic signs and symptoms after an atropine challenge strongly suggests the presence of onlichelinesterase poisoning.

DIFFERENTIAL DIAGNOSIS:

The differential diagnosis for cholinergic poisonings includes three main categories

CHOLINESTERASE INHIBITORS

Carbamate insecticides

Carbamate medicinal

Organic phosphorus insecticides

Organic phosphorus ophthalmic medications

CHOLINOMIMETICS

Aceclidine

Bethanechol

Carbachol

Methacholine

Muscarine containing mushrooms

Pilicorpine

NICOTINE ALKALOIDS

Centine

Labeline

Nicotine

Normal RBC cholinesterase levels are 5300-10,000 u/s Markedly decreased RBC cholinesterase levels are diagnostic

Depending on the RBC cholinesterase activity the levels of poisoning have been also classified into

1.Mild poisoning-activity is 20-50 percent of baseline activity

2.Moderate poisoning-activity is 10-20 percent of baseline

3.Severe poisoning-activity is <10 percent of the baseline activity.

MANAGEMENT/TREATMENT

1.PREHOSPITAL MEASURES

These include

Decontamination -done by removing all the clothing and exposed areas of the body.

This can help in removing the residual material from the skin

- Supportive care including airway centrol, oxygenation, ventilation and seizure management
- Placement of an IV line and cardiac monitoring are indicated.

2.HOSPITAL CARE

GASTRIC LAVAGE

Consider if patient presents within 60 minutes of ingestion.

Insert Nasogastric tube/gastric lavage tube

Attempt aspiration first, followed by 100 to 200 ml normal saline then aspiration

Relatively contra indicated hydrocarbon ingestion

Gastric lavage with 1:5000 KMn)4 solution

Neurologically impaired: cuffed Endotracheal tube prior to lavage.

CATHARTICS

-Used only in combination with activated charcoal

-Sorbitol

-Single dose only

-Not recommended in poisonings that produce diarrhea or those that produce ileus.

ACTIVATED CHARCOAL

Used in conjunction with a cathartic if patient presents within 60 minutes of ingestion.

Antiemetic suppository for nausea

Administered by nasogastric tube if unable to tolerate or unable to swallow.

Protect airway if hydrocarbon containing pesticide or unknown pesticide contents.

Activated charcoal should not be used routinely in management of poisoned patients-charcoal appears to be most effective within 60 minutes of ingestion and may be considered fcr use for this time period. There is insufficient evidence to support or deny its use beyond 60 minutes after ingestion.

SYRUP OF IPECAC.

-not to be administered rcutinely in poisoned patients.

ADMINISTRATION OF SPECIFIC ANTIDOTES.

Draw red cell cholinesterase and plasma pseudocholinesterase levels before therapy.

Do not delay treatment awaiting results

-Atropine administration

-Pralidoxime administration.

Atropine administration

Atropine is administered immediately to save life and continued till the achievement of the state of Atropinisation which is diagnosed by clinical manifeslation of flushing, dry mouth, papillary dilatation.

DOSE:

Adults and children> 12 years: 2 to 5 mg IV every is until pulmonary symptoms controlled.

Children < 12 years: 0.05 to 0.01 mg 1 kg every 15 min

Pralidoxime

Prolidoxime brings about anticholinergic atropine like effects resulting in freeing and reactivating chelinaterase enzymes by cleaving

phosphorylation-acetyl cholinesterase bind, it directly reads, detoxifies organophosphate molecules.

Furosemide

It is given in a dose of 40 to 160 mg IV to alleviate the pulmonary congestion remaining after full atropinisation.

Benzodiazepines:

If you give in a dose of 5 to 10 mg slow IV push for seizures, repeated every 5 to 10 mm to central or maximum upto 3c mg in adults.

Lorazepam may also be used.

Complications:

Immediate complications

- Pulmonary Oedema
- Aspiration pneumonia
- Chemical pneumonitis
- Hyper/Hypoglycemia
- Transient liver function & Coagulation abnormalities
- Death if not treated within 24 hours of poisoning

Delayed complications -complications related to CNS-

Paralysis

GBS

Precautions during treatment:

Poison arising should wear gloves, muscle etc to prevent getting persuned by absorption via skin a inhalation of poisonous fumes through respiratory tract.

Never give the following drugs:

Morphine

Aminophylline

Thenothiazine

Reserpine.

Perform blood cholinesterase level estimation to diagnose the case or assess prognosis with treatment.

AIM & OBJECTIVES

To investigate the relevance of plasma cholinesterase level to severity of OP poisoning and to evaluate usefulness of AchE in predicting clinical outcome.

MATERIALS AND METHODS

PLACE OF STUDY:

Department of Internal Medicine

Stanley Medical College.

STUDY DESIGN:

Observational Study

STUDY PERIOD:

April, 2016 to September 2016

OPERATIONAL DEFINITION:

Any person who develops muscarinic, nicotinic and CNS symptoms after dermal, respiratory or oral exposure to organophosphorus compounds such as chlorpyripos, dimethoate, sorin, tabun.

Muscarinio Symptoms	:	Salivation, lacrimation, urination, defecation, emesis, miosis, bronchorrhoca.
Microtinic Symptoms	:	Muscle weakness, Fasciculations
CNS Effects	:	Seizures, Coma, Respiratory failure.

Confirmation of OPC poisoning is based on the measurement of cholinesterase activity.

INCLUSION CRITERIA:

Patients of suspected of OPC poisoning of age > 12 years.

EXCLUSION CRITERIA

1. Patients poisoned with double insecticides.
2. Multiple poisoning with other drugs such as Opicid, diazepam, barbituarale.
3. Patients with chronic medical illness.
4. Patients on drugs such as aminophyllinc, succinyl, choline, reserpine and phenothiozinc type transquilizers.
5. Pregnant women or those who received treatment prior to admission in our hospital.

SAMPLE SIZE: 100

METHODOLOGY:

This is an observational study wherein 100 known patients of suspected OPC poisoning were evaluated clinically by history and examination. Treatment was commenced as soon as the patients were attended. The patients were given stomach wash, body was and an intravenous line was established. An intravenous bolus of atropine was administered promptly.

The patients were assessed for respiratory failure and intubated and connected to mechanical ventilation. Patients were given injection atropine till signs of atropinisation. Pralidoxime infusion were given. ABG, complete blood picture, RBS, RFT and chest X-ray were taken Serum cholinesterase level was estimated at the time of admission in all patients

Based on the serum cholinesterase levels, the severity of poisoning was defined.

HUMAN SUBJECT PROTECTION:

The full protocol along with draft questionnaire and informed consent will be kept in institutional ethical committees and approval will be obtained.

INFORMED CONSENT:

Consent form will be written in both English and Tamil and consent will be obtained from the participant's confidentiality will be maintained.

EXPECTED BENEFITS FROM STUDY:

Serum cholinesterase levels are used to assess the severity of poisoning and predicting the clinical outcome.

RESULTS AND DISCUSSION

Groups

Groups	Number
Death Group	6
Survival Group	24

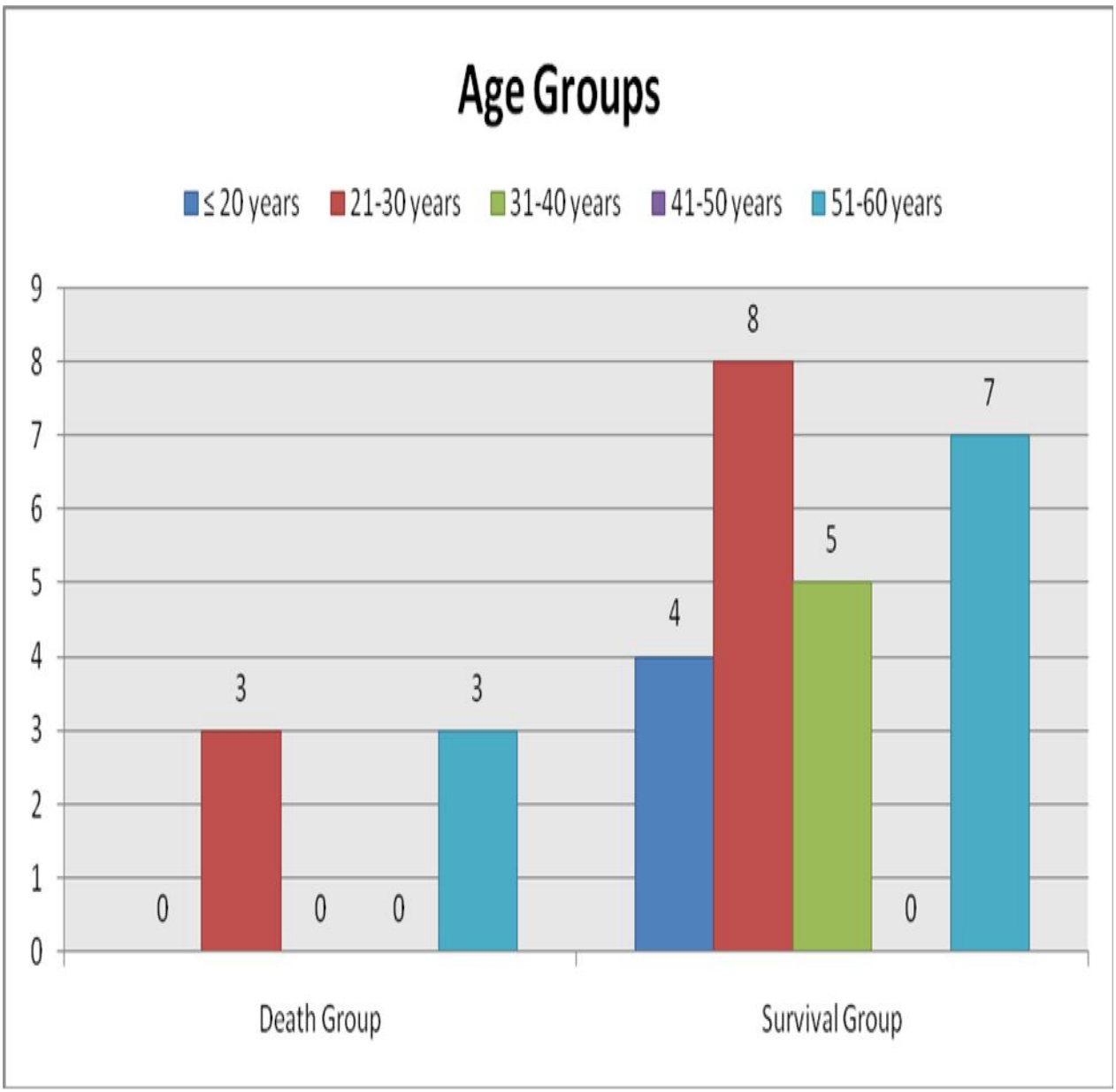
Null Hypothesis

Null Hypothesis : H0	Death group equal in effect compared to Survival group
Alternate Hypothesis : H1	Death group hazardous in effect compared to Survival group

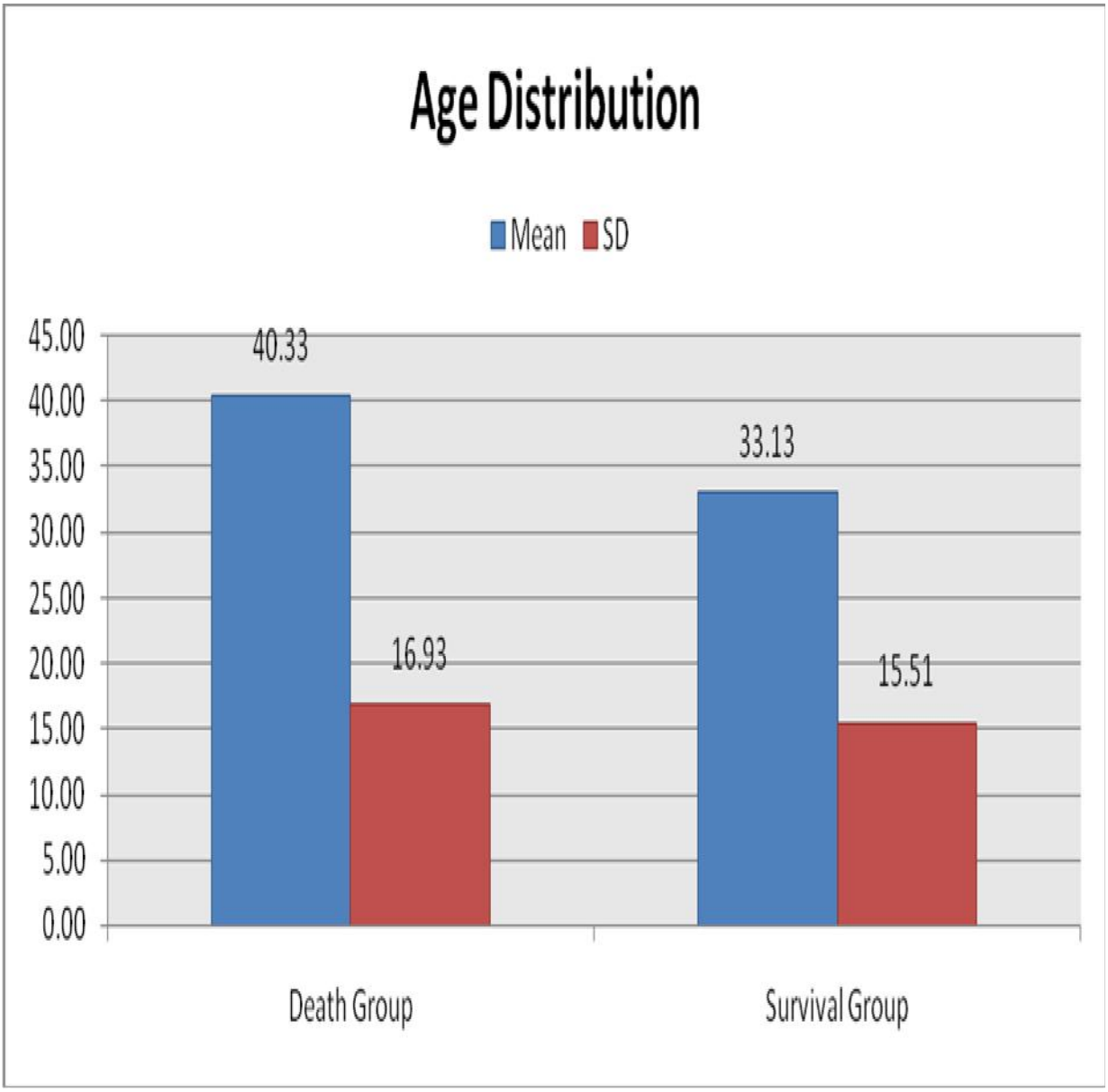
Data Analysis

Descriptive statistics was done for all data and were reported in terms of mean values and percentages. Suitable statistical tests of comparison were done. Continuous variables were analysed with the unpaired t test.. Categorical variables were analysed with the Chi-Square Test and Fisher Exact Test. Statistical significance was taken as $P < 0.05$. The data was analysed using SPSS version 16 and Microsoft Excel 2007.

Age


Age Groups
≤ 20 years
21-30 years
31-40 years
41-50 years
51-60 years
9
8
7
6
5
4
3
2
1
0
0
3
0
0
3
4
8
5
0
7
Death Group
Survival Group

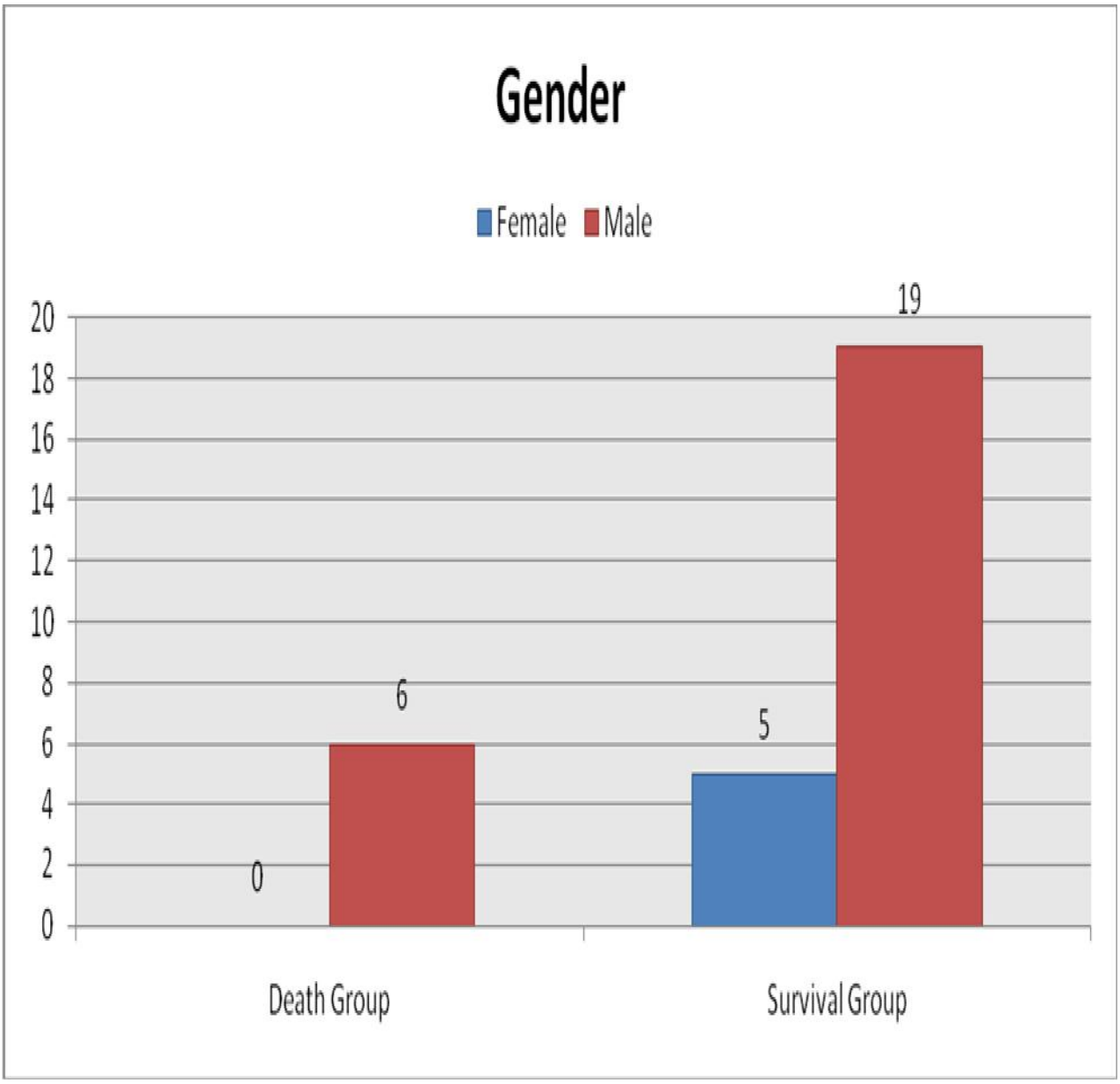
Age Groups	Death Group	Survival Group	Death Group %	Survival Group %
≤ 20 years	0	4	0.00	16.67
21-30 years	3	8	50.00	33.33
31-40 years	0	5	0.00	20.83
41-50 years	0	0	0.00	0.00
51-60 years	3	7	50.00	29.17
Total	6	24	100	100


Age Distribution
Mean
SD
45.00
40.00
35.00
30.00
25.00
20.00
15.00
10.00
5.00
0.00
40.33
16.93
33.13
15.51
Death Group
Survival Group

Age Distribution	Death Group	Survival Group
Mean	40.33	33.13
SD	16.93	15.51
P value Unpaired t Test		0.3253

In the death group majority belonged to 21-30 and 51-60 years age class interval (50.00%) with a mean age of 40.33 years. In the survival group majority belonged to 21-30 years age class interval (33.33%) with a mean age of 33.13 years. Among the study patients, there was no statistically significant difference in relation to age distribution between with death group and without survival group with a p value of >0.05 as per unpaired t test. Therefore we fail to reject the null hypothesis that there is no difference in age distribution between the study groups.

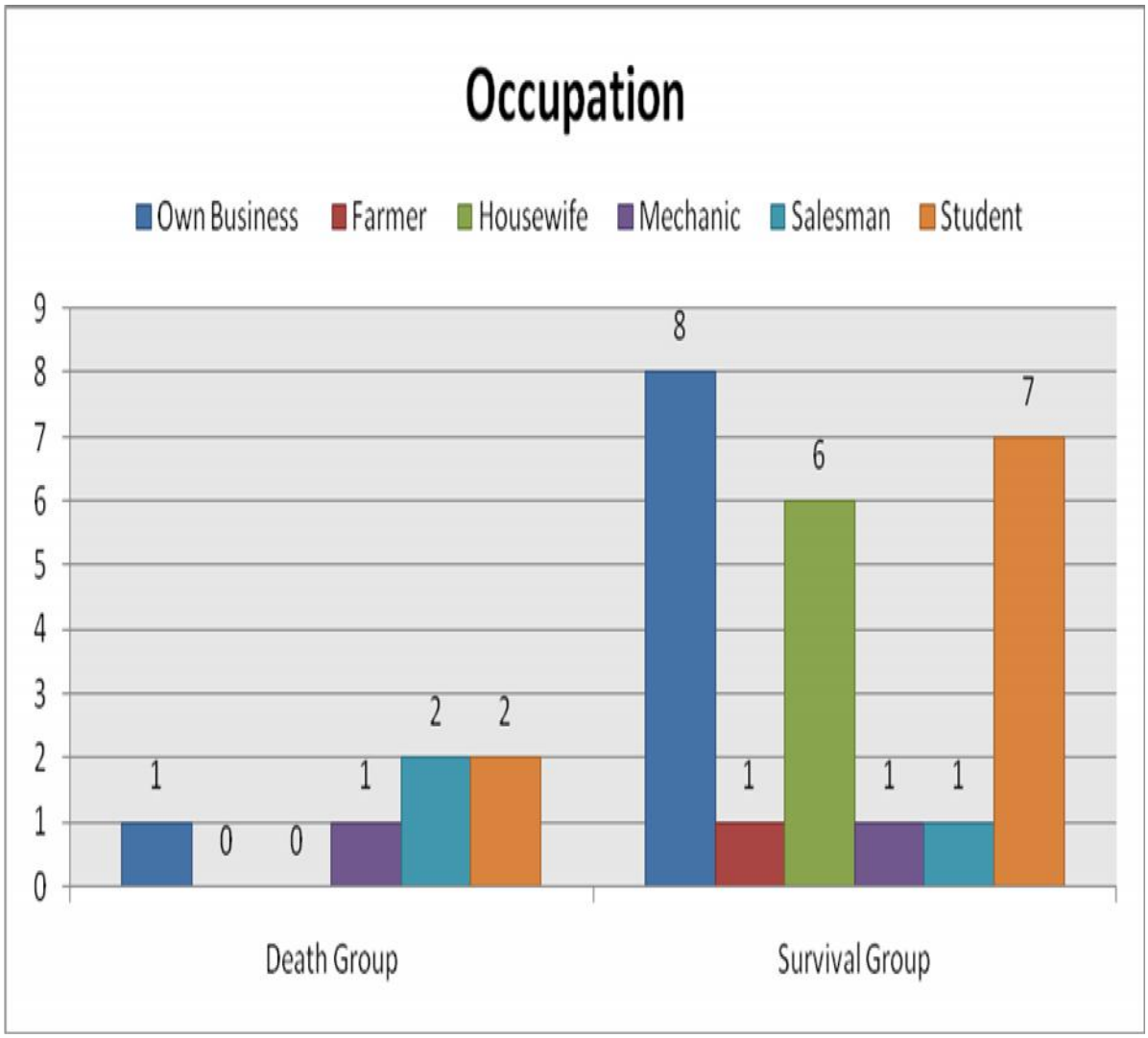
Gender


Gender
Female
Male
20
18
16
14
12
10
8
6
4
2
0
0
6
5
19
Death Group
Survival Group

Gender	Death Group	Survival Group	Death Group %	Survival Group %
Female	0	5	0.00	20.83
Male	6	19	100.00	79.17
Total	6	24	100	100
P value Fishers Exact Test			0.5526	

In the death group majority belonged to male gender class interval (100.00). In the survival group majority belonged to male gender class interval (79.17%). Among the study patients, there was no statistically significant difference in relation to gender status between with death group and without survival group with a p value of >0.05 as per fishers exact test. Therefore we fail to reject the null hypothesis that there is no difference in gender status between the study groups.

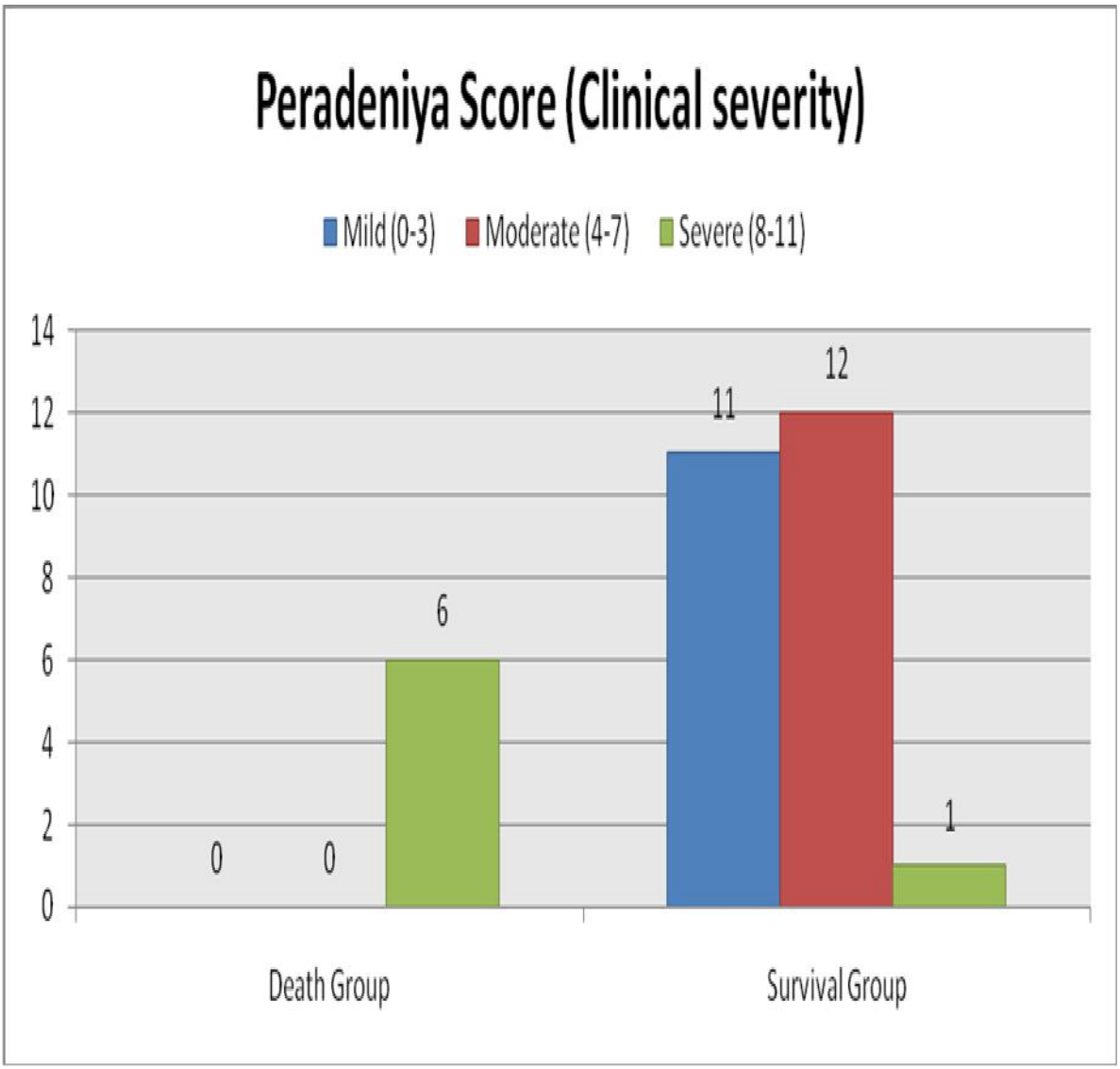
Occupation


Occupation
Own Business
Farmer
Housewife
Mechanic
Salesman
Student
9
8
7
6
5
4
3
2
1
0
1
0
0
1
2
2
8
1
6
1
1
7
Death Group
Survival Group

Occupation	Death Group	Survival Group	Death Group %	Survival Group %
Own Business	1	8	16.67	33.33
Farmer	0	1	0.00	4.17
Housewife	0	6	0.00	25.00
Mechanic	1	1	16.67	4.17
Salesman	2	1	33.33	4.17
Student	2	7	33.33	29.17
Total	6	24	100	100
P value Fishers Exact Test			0.5433	

In the death group majority belonged to salesman and student occupation class interval (33.33%). In the survival group majority belonged to own business occupation class interval (33.33%) followed by student (29.17%). Among the study patients, there was no statistically significant difference in relation to occupation status between with death group and without survival group with a p value of >0.05 as per fishers exact test. Therefore we fail to reject the null hypothesis that there is no difference in occupation status between the study groups.

Peradeniya Score (Clinical severity)



Peradeniya Score (Clinical severity)	Death Group	Survival Group	Death Group %	Survival Group %
Mild (0-3)	0	11	0.00	45.83
Moderate (4-7)	0	12	0.00	50.00
Severe (8-11)	6	1	100.00	4.17
Total	6	24	100	100
P value Fishers Exact Test			<0.0001	

In the death group majority belonged to severe category of peradaniya score class interval (100.00%). In the survival group majority belonged to moderate category of peradaniya score class interval (50.00%) followed by mild category (45.83%). Among the study patients, there was a statistically significant difference in relation to peradaniya score status between with death group and without survival group with a p value of <0.05 as per fishers exact test. Therefore we reject the null hypothesis that there is no difference in occupation status between the study groups.

Discussion

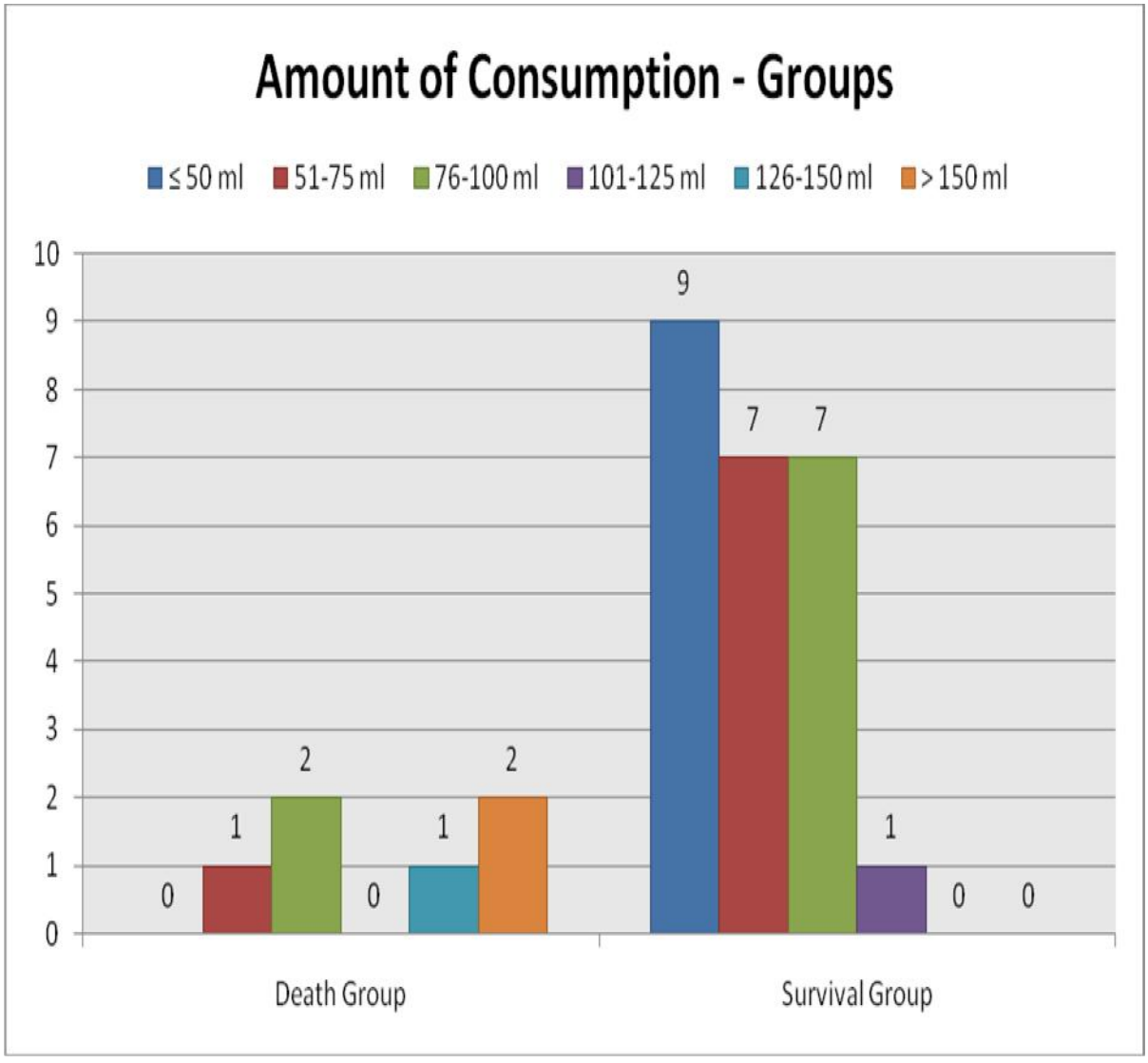
The incidence of severe category of peradaniya score was significantly more in death group compared to survival group by a percentage difference of 95.83% (96% higher). This difference is significant with a p-value of <0.0001 as per fishers exact test.

Conclusion

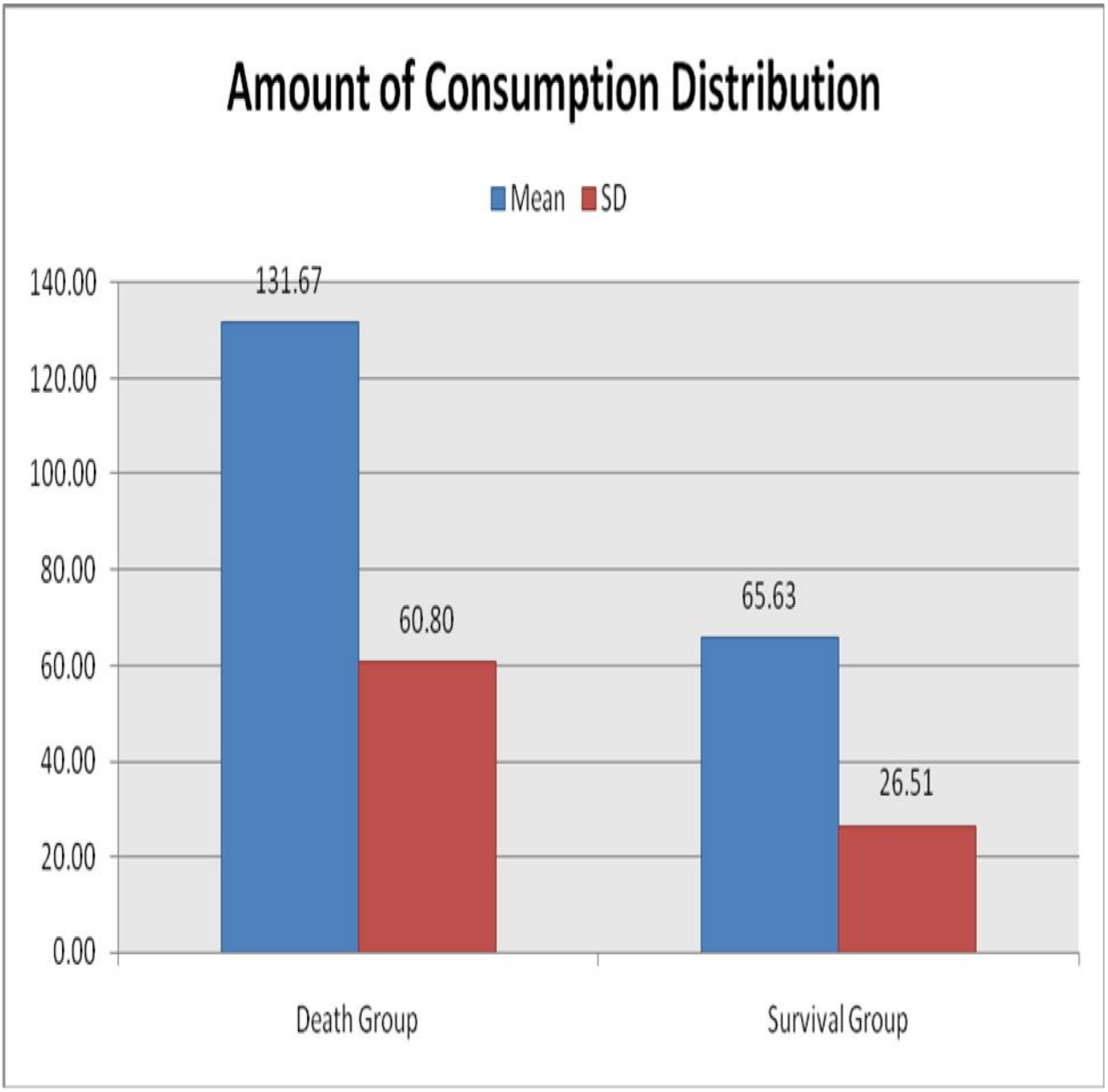
In this study we can safely conclude that a significantly increased severe category of peradaniya score incidence is associated with death group compared to increased mild category of peradaniya score incidence found in survival group in patients admitted with organophosphorus poisoning.

In other words severe category of peradaniya score occurrence is 24 times more common among patients who died due to organophosphorus poisoning compared to survived patients. The increased clinical severity in patients admitted with organophosphorus poisoning as calculated by peradaniya score results most probably in death. Hence the scale assists in grading severity of in patients admitted with organophosphorus poisoning at first contact and help in predicting possible outcome.

Amount of Consumption



Amount of Consumption - Groups	Death Group	Survival Group	Death Group %	Survival Group %
≤ 50 ml	0	9	0.00	37.50
51-75 ml	1	7	16.67	29.17
76-100 ml	2	7	33.33	29.17
101-125 ml	0	1	0.00	4.17
126-150 ml	1	0	16.67	0.00
> 150 ml	2	0	33.33	0.00
Total	6	24	100	100


Amount of Consumption Distribution
Mean
SD
140.00
120.00
100.00
80.00
60.00
40.00
20.00
0.00
131.67
60.80
65.63
26.51
Death Group
Survival Group

Amount of Consumption Distribution	Death Group	Survival Group
Mean	131.67	65.63
SD	60.80	26.51
P value Unpaired t Test		0.0003

In the death group majority belonged to 76-100 ml and > 150 ml OP consumption amount class interval (33.33%) with a mean intake of 131.67 ml. In the survival group majority belonged to < 50 ml OP consumption amount class interval (65.63%) followed by 51-75 ml and 76-100 ml class interval (29.17%) with a mean intake of 65.63 ml. Among the study patients, there was a statistically significant difference in relation to OP consumption amount distribution between with death group and without survival group with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in OP consumption amount distribution between the study groups.

Discussion

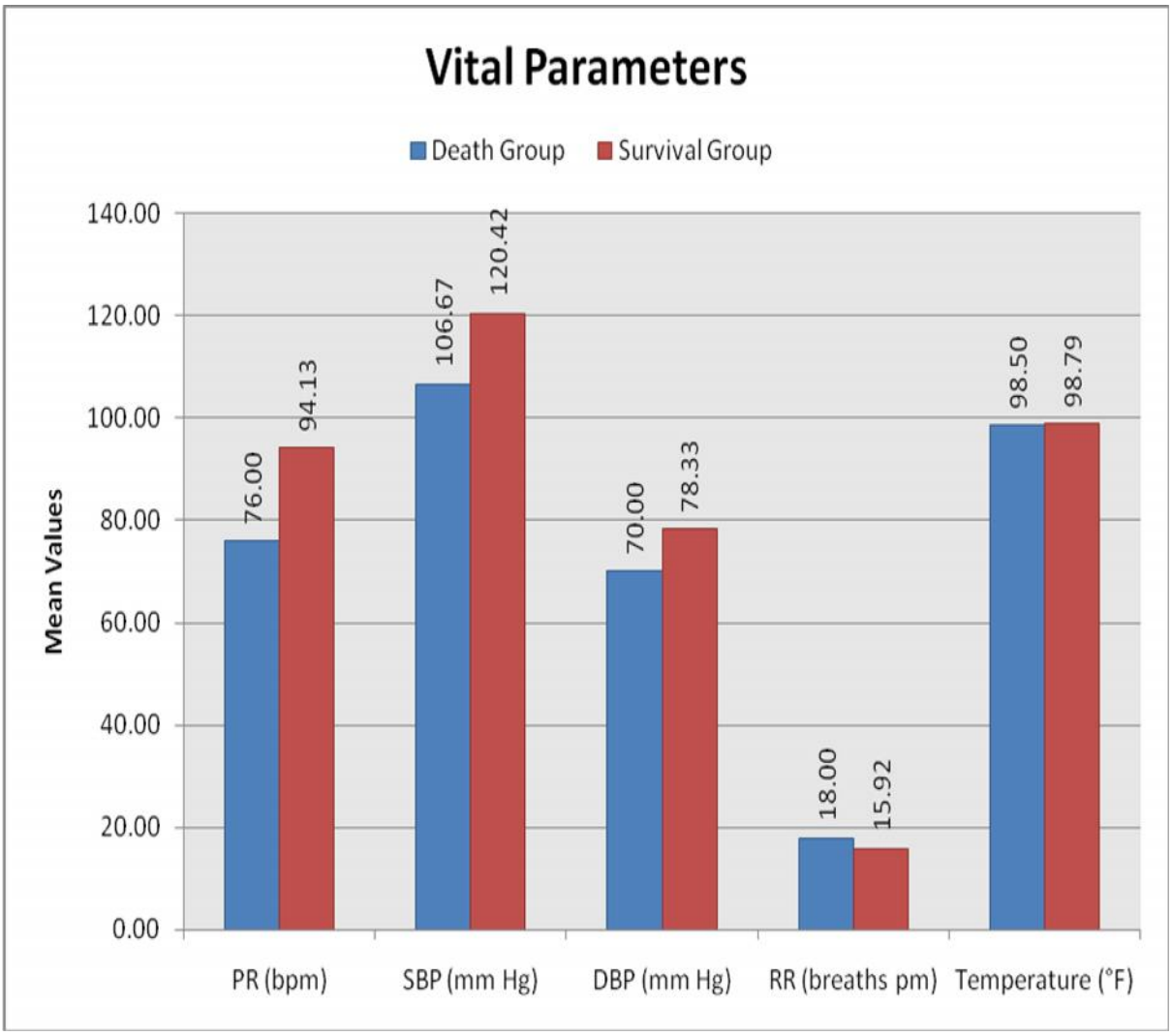
The mean OP consumption amount was significantly more in death group compared to survival group by a mean difference of 66.04 ml(50.00% higher). This difference is significant with a p-value of 0.0003 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significantly increased mean OP consumption amount is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

In other words mean OP consumption amount is 2.01 times more among patients who died due to organophosphorus poisoning compared to survived patients. The increased mean OP consumption amount in patients admitted with organophosphorus poisoning results most probably in death especially if it is more than 125 ml. Hence mortality is directly proportionate to the amount of OP substances consumed.

Vital Parameters



Vital Parameters		PR (bpm)	SBP (mm Hg)	DBP (mm Hg)	RR (breaths pm)
Death Group	Mean	76.00	106.67	70.00	18.00
	SD	2.19	5.16	0.00	4.38
Survival Group	Mean	94.13	120.42	78.33	15.92
	SD	16.20	13.01	7.61	3.36
P value Unpaired t Test		0.0117	0.0181	0.0132	0.2109

In the death group the mean pulse rate, systolic blood pressure, diastolic blood pressure, respiratory rate and temperature was 76.00, 106.67, 70.00, 18.00 and 98.50 respectively. Similarly in survival group the mean pulse rate, systolic blood pressure, diastolic blood pressure, respiratory rate and temperature was 94.13, 102.42, 78.33, 15.92 and 98.79 respectively. Among the study patients, there was a statistically significant difference in relation to PR, SBP and DBP distribution between with death group and without survival group with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in PR, SBP and DBP distribution between the study groups.

Discussion

The mean PR was significantly less in death group compared to survival group by a mean difference of 18.13 bpm (19.00% lower). This difference is significant with a p-value of 0.0117 as per unpaired t test.

The mean SBP was significantly less in death group compared to survival group by a mean difference of 13.75 mm Hg (11.00% lower). This difference is significant with a p-value of 0.0181 as per unpaired t test.

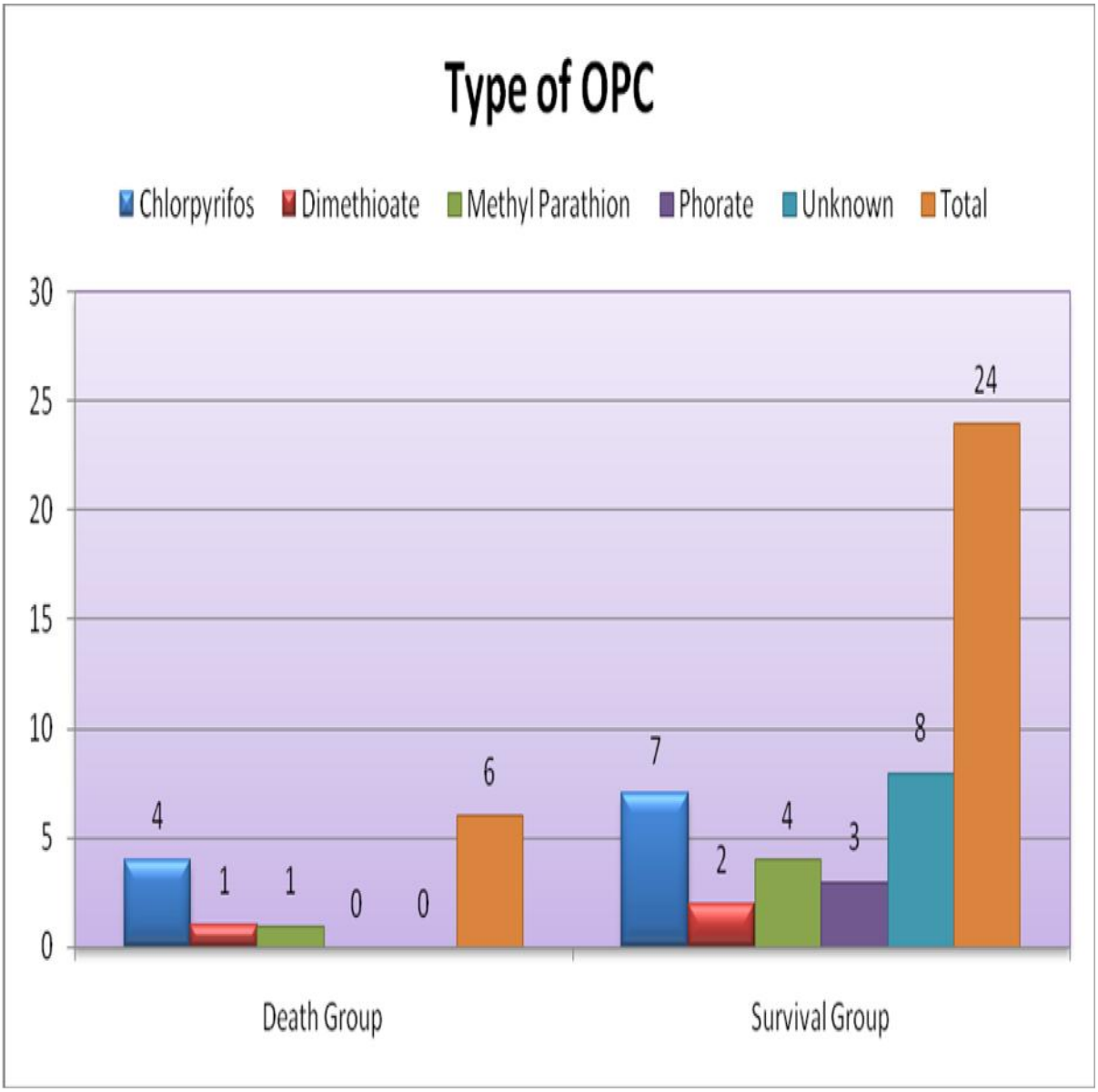
The mean DBP was significantly less in death group compared to survival group by a mean difference of 8.33 mm Hg (11.00% lower). This difference is significant with a p-value of 0.0132 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significantly decreased PR, SBP and DBP is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

The mean PR, SBP and DBP are 1.24, 1.13 and 1.12 times more among patients who died due to organophosphorus poisoning compared to survived patients. The increased mean PR, SBP and DBP serves as a risk factor for death in patients admitted with organophosphorus poisoning.

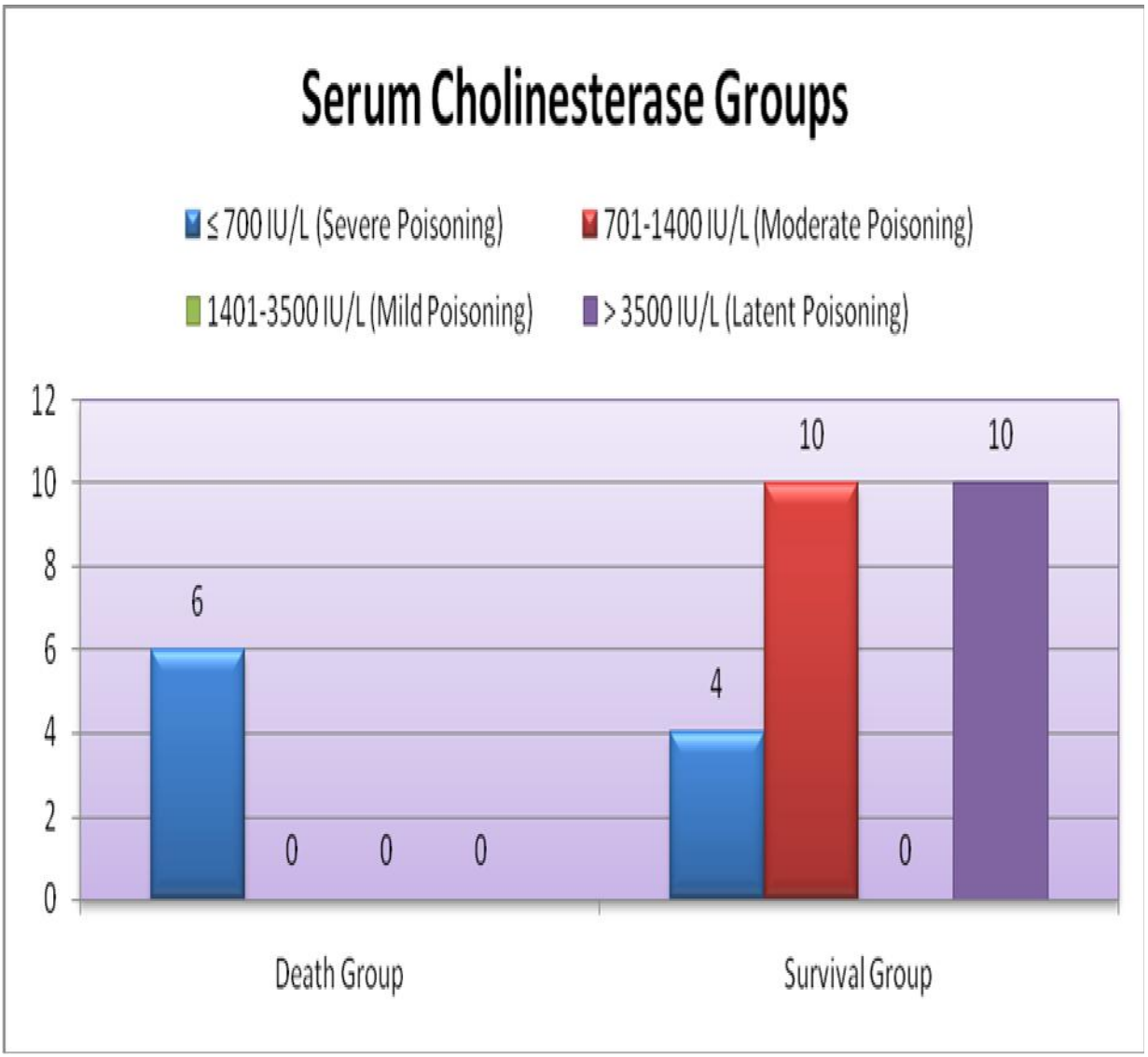
Type of OPC


Type of OPC
Chlorpyrifos
Dimethioate
Methyl Parathion
Phorate
Unknown
Total
30
25
20
15
10
5
0
4
1
1
0
0
6
7
2
4
3
8
24
Death Group
Survival Group

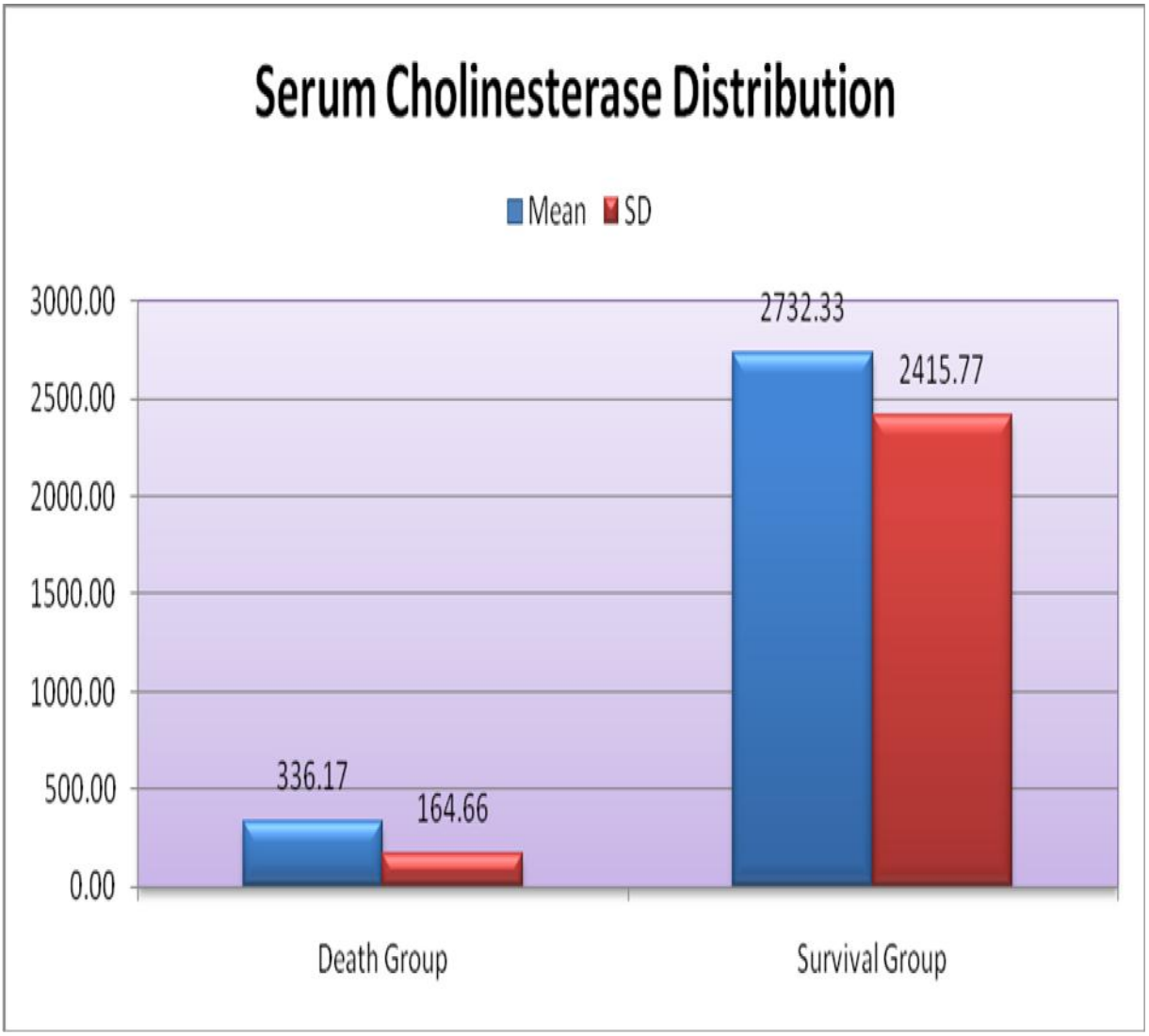
Type of OPC	Death Group	Survival Group	Death Group %	Survival Group %
Chlorpyrifos	4	7	66.67	29.17
Dimethioate	1	2	16.67	8.33
Methyl Parathion	1	4	16.67	16.67
Phorate	0	3	0.00	12.50
Unknown	0	8	0.00	33.33
Total	6	24	100	100
P value Fishers Exact Test			0.0843	

In the death group majority consumed chlorpyrifos (66.67%) followed by dimethionate and methyl parathion (16.67%). In the survival group majority consumed chlorpyrifos (29.17%) followed by methyl parathion (16.67%). Among the study patients, there was no statistically significant difference in relation to type of OPC consumed between with death group and without survival group with a p value of >0.05 as per fishers exact test. Therefore we fail to reject the null hypothesis that there is no difference in type of OPC consumed between the study groups.

Serum Cholinesterase



Serum Cholinesterase Groups	Death Group	Survival Group	Death Group %	Survival Group %
≤ 700 IU/L (Severe Poisoning)	6	4	100.00	16.67
701-1400 IU/L (Moderate Poisoning)	0	10	0.00	41.67
1401-3500 IU/L (Mild Poisoning)	0	0	0.00	0.00
> 3500 IU/L (Latent Poisoning)	0	10	0.00	41.67
Total	6	24	100	100


Serum Cholinesterase Distribution
Mean
SD
3000.00
2500.00
2000.00
1500.00
1000.00
500.00
0.00
336.17
164.66
2732.33
2415.77
Death Group
Survival Group

Serum Cholinesterase Distribution	Death Group	Survival Group
Mean	336.17	2732.33
SD	164.66	2415.77
P value Unpaired t Test		0.0235

In the death group majority belonged to ≤ 700 IU/L (severe poisoning) serum cholinesterase level class interval (100.00%) with a mean intake of 336.17 IU/L. In the survival group majority belonged to > 3500 IU/L (latent poisoning) serum cholinesterase level class interval (41.67%) with a mean intake of 2732.33 IU/L. Among the study patients, there was a statistically significant difference in relation to serum cholinesterase level distribution between with death group and without survival group with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in serum cholinesterase level distribution between the study groups.

Discussion

The mean serum cholinesterase level was significantly less in death group compared to survival group by a mean difference of 2396.17 IU/L

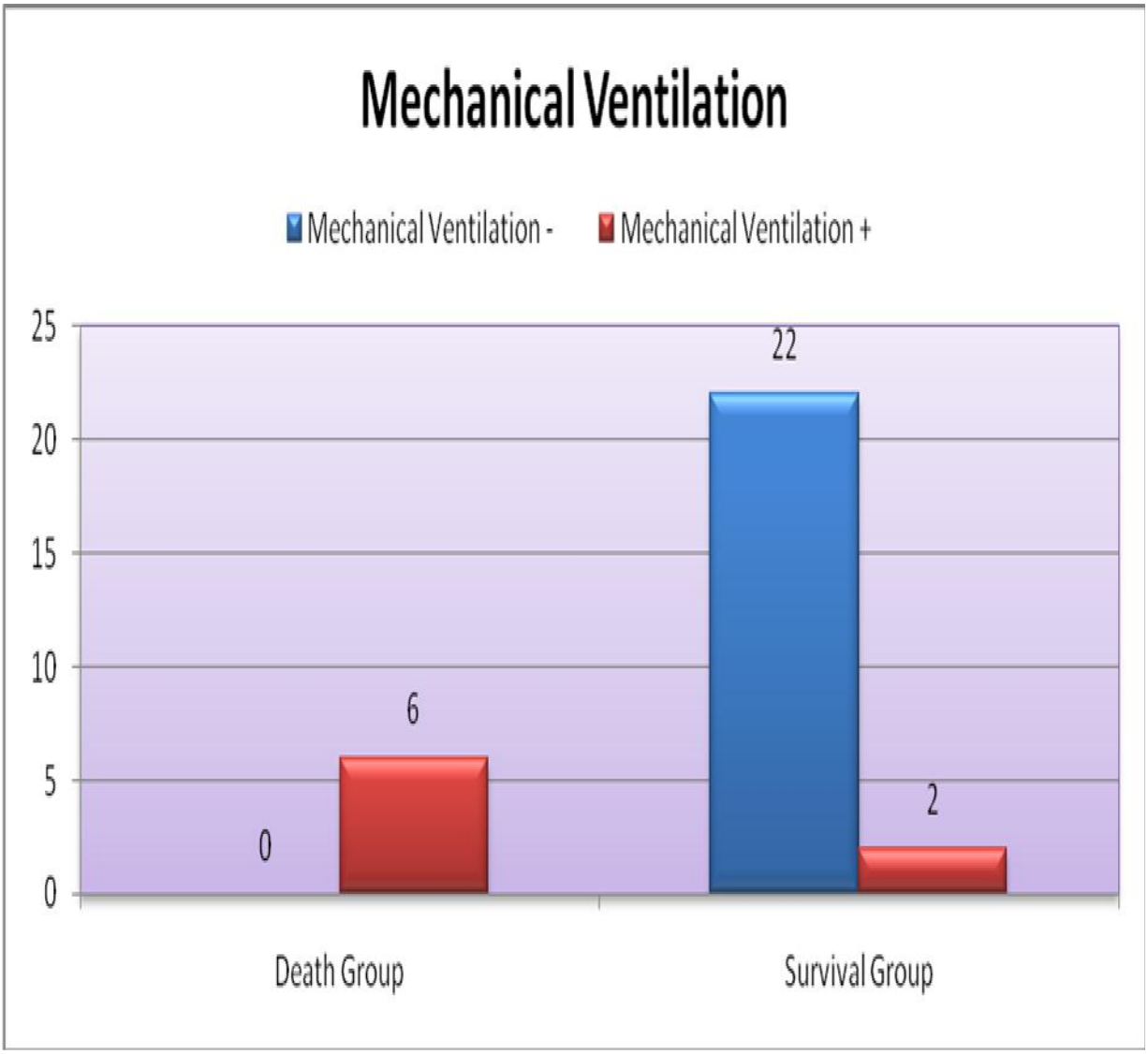
(88.00% lower). This difference is significant with a p-value of 0.0235 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significantly decreased mean serum cholinesterase level is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

In other words extremely lowered mean serum cholinesterase level is 8.13 times more among patients who died due to organophosphorus poisoning compared to survived patients. The decreased mean serum cholinesterase level in patients admitted with organophosphorus poisoning results most probably in death especially if it is less than ≤ 700 IU/L. Hence mortality is inversely proportionate to the mean serum cholinesterase level measured in the patient on admission.

Mechanical Ventilation



Mechanical Ventilation	Death Group	Survival Group	Death Group %	Survival Group %
Mechanical Ventilation -	0	22	0.00	91.67
Mechanical Ventilation +	6	2	100.00	8.33
Total	6	24	100	100
P value Fishers Exact Test			<0.0001	

In the death group majority had mechanical ventilation (100.00%). In the survival group majority had no mechanical ventilation (91.67%). Among the study patients, there was a statistically significant difference in relation to mechanical ventilation status between with death group and without survival group with a p value of <0.05 as per fishers exact test. Therefore we reject the null hypothesis that there is no difference in mechanical ventilation status between the study groups.

Discussion

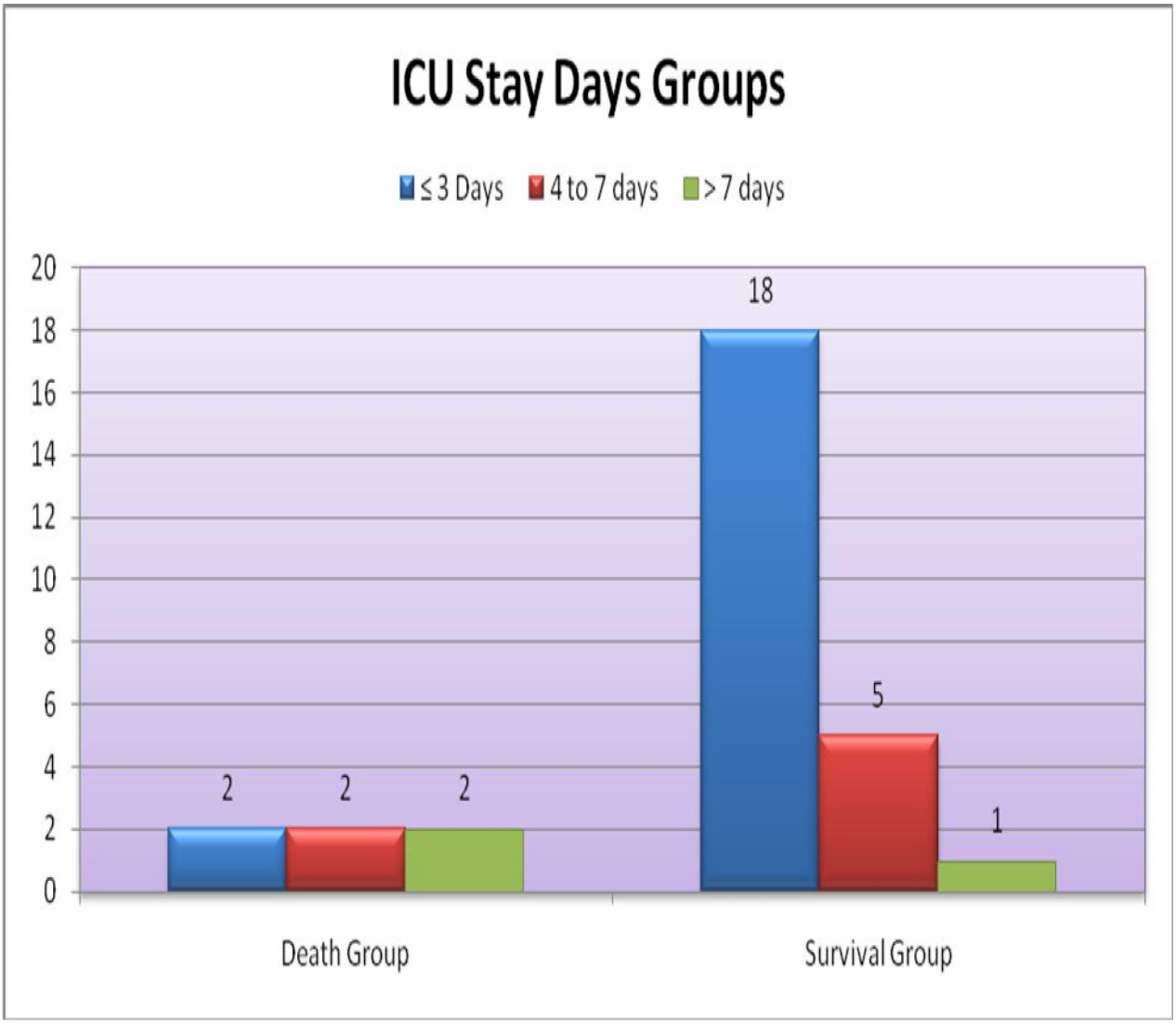
The incidence of mechanical ventilation was significantly more in death group compared to survival group by a percentage difference of 91.67% (92% higher). This difference is significant with a p-value of <0.0001 as per fishers exact test.

Conclusion

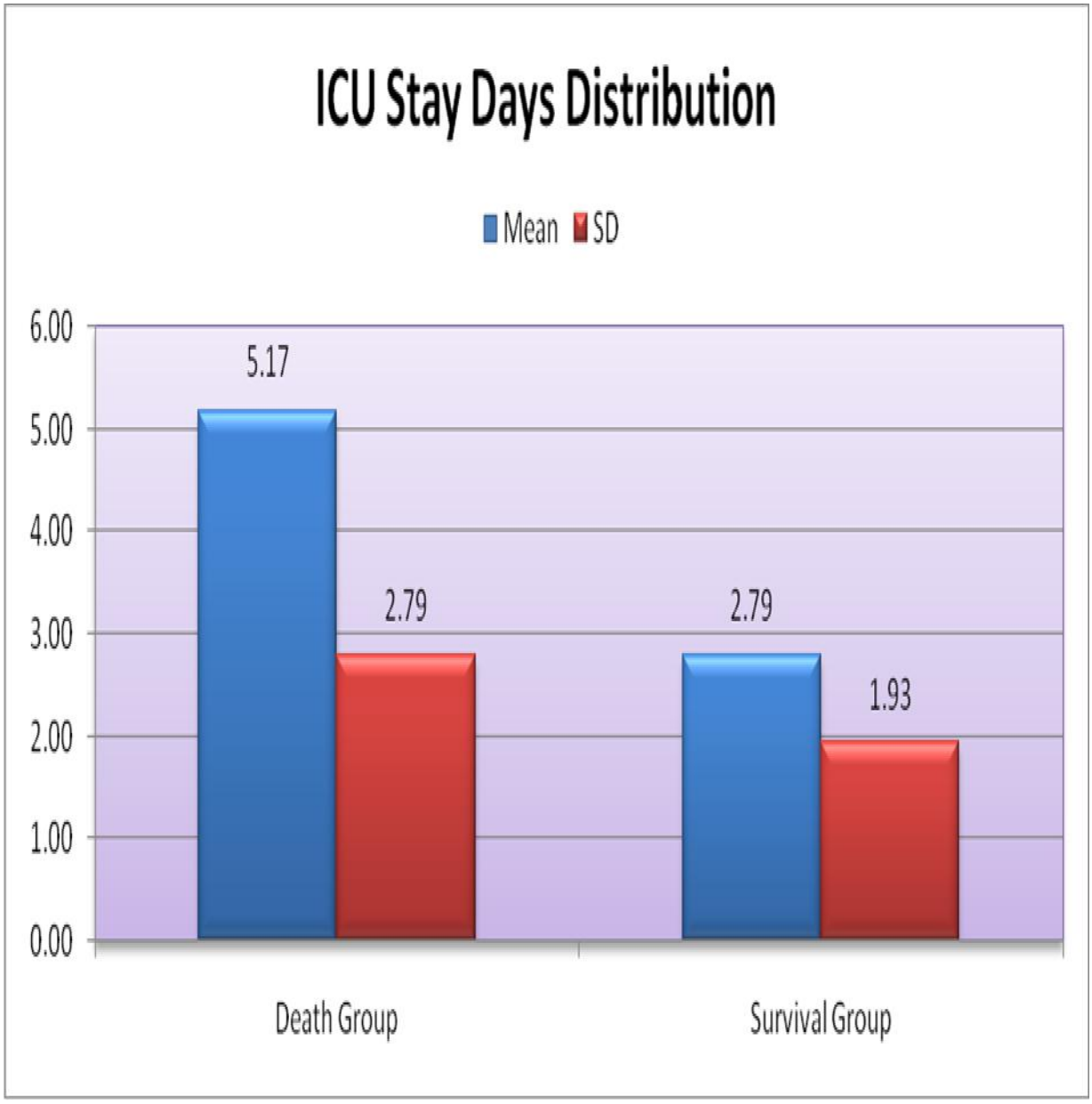
In this study we can safely conclude that a significantly increased mechanical ventilation incidence is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

In other words mechanical ventilation occurrence is 12 times more common among patients who died due to organophosphorus poisoning compared to survived patients. hence mechanical ventilation in patients admitted with organophosphorus poisoning is a risk factor for predicting mortality and most often results in death.

ICU Stay Days



ICU Stay Days Groups	Death Group	Survival Group	Death Group %	Survival Group %
≤ 3 Days	2	18	33.33	75.00
4 to 7 days	2	5	33.33	20.83
> 7 days	2	1	33.33	4.17
Total	6	24	100	100


ICU Stay Days Distribution
Mean
SD
6.00
5.00
4.00
3.00
2.00
1.00
0.00
5.17
2.79
2.79
1.93
Death Group
Survival Group

ICU Stay Days Distribution	Death Group	Survival Group
Mean	5.17	2.79
SD	2.79	1.93
P value Unpaired t Test		0.0201

In the death group majority belonged to > 7 days ICU stay duration (33.33%) with a mean stay of 5.17 days. In the survival group majority belonged to ≤ 3 Days ICU stay duration (41.67%) with a mean stay of 2.79 days. Among the study patients, there was a statistically significant difference in relation to ICU stay duration between with death group and without survival group with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in ICU stay duration between the study groups.

Discussion

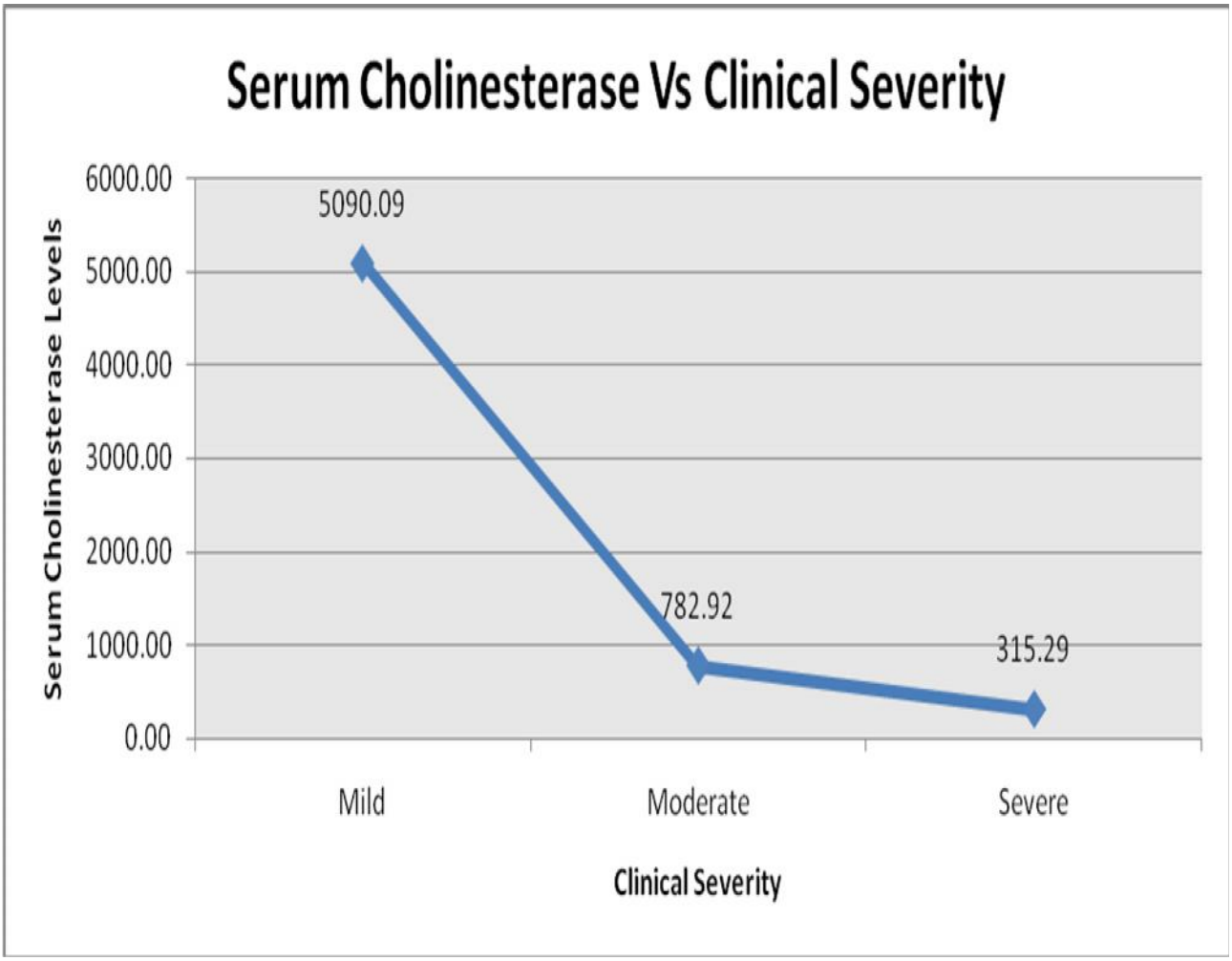
The mean ICU stay duration was significantly more in death group compared to survival group by a mean difference of 2.38 days (46.00% higher). This difference is significant with a p-value of 0.0201 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significantly increased mean ICU stay duration is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

In other words ICU stay duration is elevated 1.85 times more among patients who died due to organophosphorus poisoning compared to surviving patients. The increased mean ICU stay duration in patients admitted with organophosphorus poisoning results most probably in death especially if it is greater than 5 days. Hence mortality is directly proportionate to the mean ICU stay duration.

Serum Cholinesterase Vs Clinical Severity



Serum Cholinesterase Vs Clinical Severity	Mild	Moderate	Severe
Mean	5090.09	782.92	315.29
SD	1422.07	267.68	160.14
P value Single Factor ANOVA			<0.0001

When serum cholinesterase levels were matched against clinical severity, the mild , moderate and severe category of clinical severity patients had mean serum cholinesterase levels of 5090.09, 782.92 and 315.29 IU/L respectively. Among the study patients, there was a statistically significant difference in relation to serum cholinesterase levels between clinical severity groups with a p value of <0.05 as per single factor ANOVA test. Therefore we reject the null hypothesis that there is no difference in serum cholinesterase levels between clinical severity groups.

Discussion

The mean serum cholinesterase levels were significantly more in mild clinical severity group compared to moderate clinical severity group by a mean difference of 4307.17 IU/L (85.00% higher).

The mean serum cholinesterase levels were significantly more in moderate clinical severity group compared to severe clinical severity group by a mean difference of 467.63 IU/L (60.00% higher).

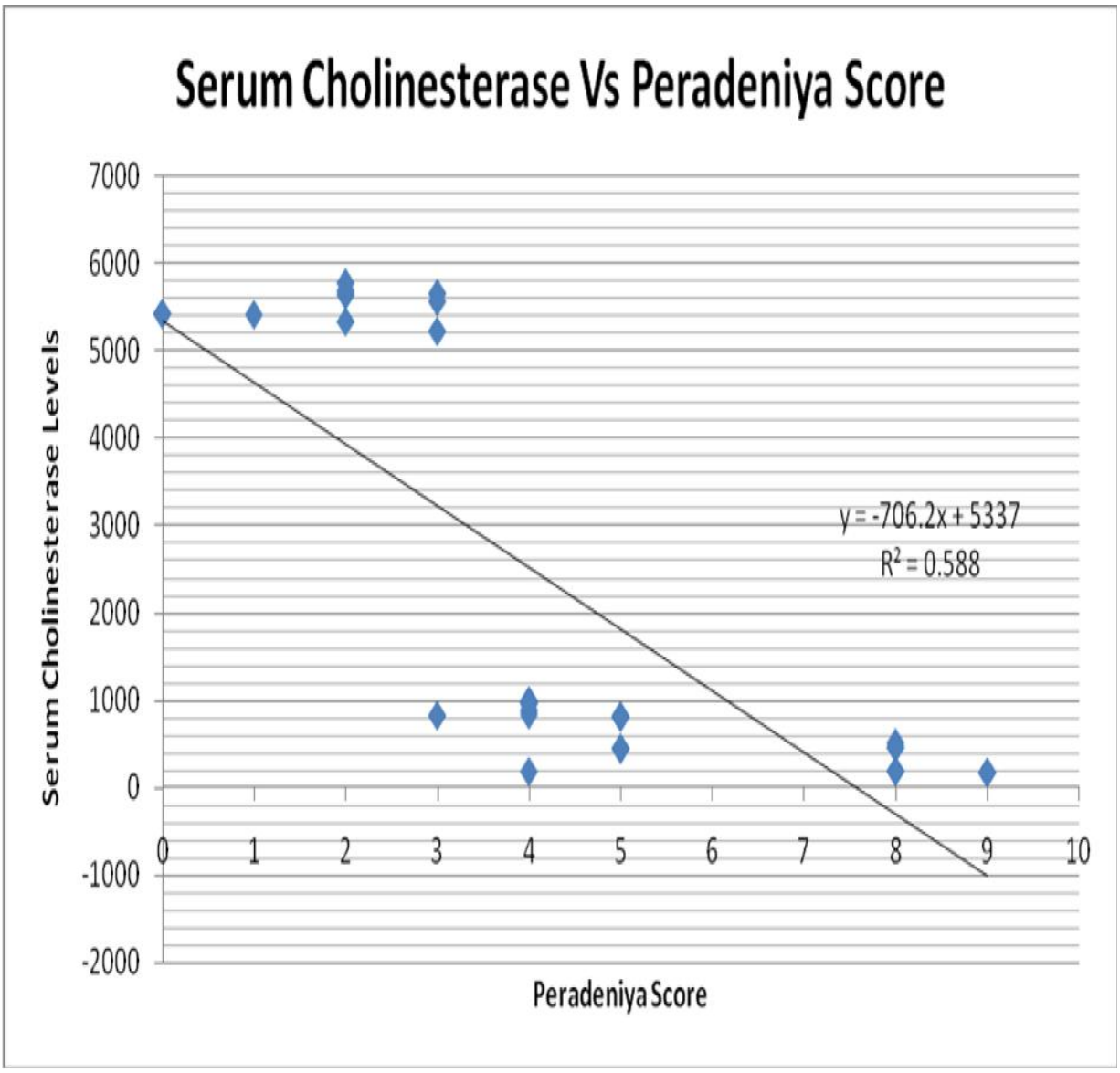
This difference is significant with a p-value of< 0.0001 as per single factor ANOVA test.

Conclusion

In this study we can safely conclude that a significantly increased mean serum cholinesterase levels is associated with mild clinical severity group compared to significantly decreased mean serum cholinesterase levels is associated with severe clinical severity group in patients admitted with organophosphorus poisoning.

In other words mean serum cholinesterase levels is elevated 6.50 times more among mild clinical severity group patients compared to severe clinical severity group patients admitted with organophosphorus poisoning. Hence clinical severity is inversely proportionate to the mean serum cholinesterase levels.

Serum Cholinesterase Vs Peradeniya Score



Serum Cholinesterase Vs Peradeniya Score - Correlation and Regression Statistics	
Multiple R	-0.766964
R Square	0.588233
Adjusted R Square	0.573527
Standard Error	1.675812
Observations	30
Significance F	<0.0001

There is a strong negative and inverse correlation between increase in serum cholinesterase levels and corresponding decrease in *peradeniya score*. This is indicated by the Pearson's R Correlation value of -0.766964. This means as serum cholinesterase levels increases the *peradeniya score* decreases. As per Pearson's R Correlation this increase in serum cholinesterase levels due to corresponding increase in *peradeniya score* happens 77% of times.

This direct negative, inverse and high correlation is significant with a p value of <0.0001 as per analysis of variance test. The percentage change is also explained in the scatter plot. This linear model explains all the variability of the response data around its mean. Since $R^2$is 0.588233,

“the fitted regression equation explains 59% of the variation in Y"(Y= -706.23 (perdeniya score) + 1099).

Thus 1 score point increase in *peradeniya score* causes 392.77 IU/L increase in in serum cholinesterase levels. This variation in serum cholinesterase levels in relation to *peradeniya score* correlates 77% of times and this variation is truly accounted 59% of times.

So we can conclude that elevated serum cholinesterase levels is an independent negative, inverse and strong correlate of *peradeniya score* in patients with type 2 diabetes mellitus.

Limitations

1. Low sample size. So little space for robust statistical analysis.
2. Study population restricted to patients referred to our department
3. Selection bias may have influenced the results.
4. Poor financial support
5. Inability to use research design like cohort study due to paucity of time and resources

CONCLUSION

In this study we can safely conclude that a significantly increased severe category of peradaniya score incidence is associated with death group compared to increased mild category of peradaniya score incidence found in survival group in patients admitted with organophosphorus poisoning.

In other words severe category of peradaniya score occurrence is 24 times more common among patients who died due to organophosphorus poisoning compared to survived patients. The increased clinical severity in patients admitted with organophosphorus poisoning as calculated by peradaniya score results most probably in death. Hence the scale assists in grading severity of in patients admitted with organophosphorus poisoning at first contact and help in predicting possible outcome.

In this study we can safely conclude that a significantly increased mean OP consumption amount is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

In other words mean OP consumption amount is 2.01 times more among patients who died due to organophosphorus poisoning compared

to survived patients. The increased mean OP consumption amount in patients admitted with organophosphorus poisoning results most probably in death especially if it is more than 125 ml. Hence mortality is directly proportionate to the amount of OP substances consumed.

In this study we can safely conclude that a significantly decreased PR, SBP and DBP is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

The mean PR, SBP and DBP are 1.24, 1.13 and 1.12 times more among patients who died due to organophosphorus poisoning compared to survived patients. The increased mean PR, SBP and DBP serves as a risk factor for death in patients admitted with organophosphorus poisoning.

In this study we can safely conclude that a significantly decreased mean serum cholinesterase level is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

In other words extremely lowered mean serum cholinesterase level is 8.13 times more among patients who died due to organophosphorus poisoning compared to survived patients. The decreased mean serum cholinesterase level in patients admitted with organophosphorus

poisoning results most probably in death especially if it is less than ≤ 700 IU/L. Hence mortality is inversely proportionate to the mean serum cholinesterase level measured in the patient on admission.

In this study we can safely conclude that a significantly increased mechanical ventilation incidence is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

In other words mechanical ventilation occurrence is 12 times more common among patients who died due to organophosphorus poisoning compared to survived patients. hence mechanical ventilation in patients admitted with organophosphorus poisoning is a risk factor for predicting mortality and most often results in death.

In this study we can safely conclude that a significantly increased mean ICU stay duration is associated with death group compared to survival group in patients admitted with organophosphorus poisoning.

In other words ICU stay duration is elevated 1.85 times more among patients who died due to organophosphorus poisoning compared to surviving patients. The increased mean ICU stay duration in patients admitted with organophosphorus poisoning results most probably in death

especially if it is greater than 5 days. Hence mortality is directly proportionate to the mean ICU stay duration.

In this study we can safely conclude that a significantly increased mean serum cholinesterase levels is associated with mild clinical severity group compared to significantly decreased mean serum cholinesterase levels is associated with severe clinical severity group in patients admitted with organophosphorus poisoning.

In other words mean serum cholinesterase levels is elevated 6.50 times more among mild clinical severity group patients compared to severe clinical severity group patients admitted with organophosphorus poisoning. Hence clinical severity is inversely proportionate to the mean serum cholinesterase levels.

SUMMARY

OP Poisoning is a major public health problem in the developing world. Clinical manifestations following OP poisoning can be associated with the extent of decrease of Pche.

This study was undertaken to find out the relationship between OPC poisoning and serum cholinesterase levels

Cholinesterase is an enzyme which is responsible for breakdown of Acetyl Choline.

OPC compounds being anticholinesterases increases acetyl choline level in the synaptic cleft thereby producing clinical toxicity.

All patients admitted with OPC poisoning are given Atropine and Pralidoxime. However it was found in various studies that serum cholinesterase level does not correlate with total dose of Atropine and hence has no prognostic value in various organophosphorus poisoning.

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PROFORMA

NAME: AGE: SEX:

ADDRESS: CONTACT NO:

OCCUPATION :

COMPLAINTS:

PAST H/O

DIABETES: 1. Yes 2. No If yes specify_____

HYPERTENSION 1. Yes 2. No If yes specify_____

CARDIAC ILLNESS 1. Yes 2. No If yes specify_____

CKD 1.Yes 2.No If yes specify

PERSONAL H/O:

H/O SMOKING:

H/O ALCOHOL:

1. Duration:

2. Frequency of intake:

3.Quantity:

FAMILY HISTORY:

RELEVANT CLINICAL EXAMINATION

BP: PR: RR:

CVS: RS:

PA:

CNS:

DATA OF MORBIDITY AND MORTALITY:

Features	Present/Absent	PChe – Day 1	PChe – Day 5
O2 Therapy with mask			
Respiratory Distress			
Ventilatory Support			
Survived			
Died			

PLASMA CHOLINESTERASE LEVELS:

Features	Present/Absent	PChe – Day 1
Latent	**3,500 IU/L**	
Mild Poisoning	**1,401 – 3500 IU/L**	
Moderate poisoning	**701 – 1,400 IU/L**	
Severe Poisoning	**<700 IU/L**	

GOVT. STANLEY MEDICAL COLLEGE, CHENNAI – 600001

INFORMED CONSENT

"OPC POISONING AND SERUM CHOLINESTERASE LEVELS" AT GOVERNMENT STANLEY MEDICAL COLLEGE HOSPITAL, CHENNAI.

Place of study: Govt. Stanley medical college, Chennai

I ……. have been informed about the details of the study in my own language.

I am aware that am suffering from decompensated disease of the liver and am willing to participate in this study where my serum sodium levels will be measured for further correlation with my health condition.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I agree to collect samples of blood/saliva/urine/tissue if study needs.

I understand that I can withdraw from the study at any point of time and even then, I can receive the medical treatment as usual.

I understand that I will not get any money for taking part in the study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full cooperation for this study.

Volunteer:	Witness:
Name and address	Name and address
Signature/thumb impression:	Signature/thumb impression
Date:	Date:

Investigator Signature and date

GOVT. STANLEY MEDICAL COLLEGE, CHENNAI – 600001

INFORMED CONSENT

“OPC POISONING AND SERUM CHOLINESTERASE LEVELS”

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ABBREVIATIONS

Ache	-	Acetyl Cholinesterase
chE	-	Cholinesterase
BuchE	-	Butyryl Cholinesterase
RBC	-	Red Blood Cell

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : OPC Poisoning and serum Cholinesterase level.

Principal Investigator : Dr. M Owaise Ahmed

Designation : PG M D (General Medicine)

Department : Department of General Medicine,
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 24.03.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY, 29/3/16.
IEC, SMC, CHENNAI